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Imaging the spontaneous obliteration of a cerebral arteriovenous malformation using c-arm cone beam computed tomography: A case report

Maria Braileanu, Wuyang Yang¹, Justin M. Caplan¹, Judy Huang¹

School of Medicine, Georgetown University, Washington, DC 20057, ¹Department of Neurosurgery, Johns Hopkins University School of Medicine, Baltimore, Maryland 21287, USA

E-mail: Maria Braileanu - mbraile1@yahoo.com; WuyangYang - wyang19@jhmi.edu; Justin M. Caplan - justincaplan@jhmi.edu; *Judy Huang - jhuang24@jhmi.edu *Corresponding author

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Abstract

Background: Spontaneous occlusion of a cerebral arteriovenous malformation (AVM) without treatment is a rare occurrence.

Case Description: We report the case of a 56-year-old male who presented with aphasia and right hemiparesis secondary to intracerebral and intraventricular hemorrhage. Diagnostic digital subtraction angiography (DSA) and c-arm cone beam computed tomography (CBCT) demonstrated a 5 mm Spetzler-Martin Grade III left thalamic AVM drained by the internal cerebral vein. Subsequent DSA and CBCT studies confirmed the spontaneous obliteration of the AVM.

Conclusions: In this case, CBCT provided high resolution imaging of the AVM. Future clinical use of CBCT as an adjunct to DSA may enhance the diagnostic and therapeutic imaging of vascular lesions.

Key Words: Arteriovenous malformation, c-arm cone beam computed tomography, digital subtraction angiography



INTRODUCTION

The spontaneous occlusion of an intracranial arteriovenous malformation (AVM) without treatment occurs rarely, with an estimated incidence of 0.5–1.3%.^[1,2,4,5] Although the exact causes of this phenomenon are unknown, regression and obliteration of AVMs have been associated with altered intracranial hemodynamics.^[1,2,4,5]

Digital subtraction angiography (DSA) is currently considered the gold-standard imaging modality for characterizing AVMs. Recently, c-arm cone beam computed tomography (CBCT) has emerged as a clinically useful technology in producing cross-sectional images of AVMs.^[6,7,9] This study describes the case of a spontaneously occluded AVM imaged with both DSA and CBCT.

CASE REPORT

A 56-year-old male initially presented with aphasia and right hemiparesis. Head CT, and brain magnetic resonance imaging (MRI) revealed a left thalamic intracerebral hemorrhage with intraventricular extension [Figure 1]. No AVM was detected on MR angiography (MRA). Given the high suspicion for an underlying vascular lesion, DSA was performed, which revealed enlargement of the left anterior

SNI: Stereotactic 2015, Vol 6, Suppl 15 - A Supplement to Surgical Neurology International

choroidal artery and early drainage into the left internal cerebral vein with no discernible nidus, likely due to the concurrent hematoma [Figure 2a]. The patient recovered his language and motor function over the next 3 months.

Diagnostic DSA and CBCT performed 3 months after presentation demonstrated a Spetzler-Martin Grade III left thalamic AVM [Figure 2b, d and e]. The 5 mm nidus was predominantly supplied by the left anterior choroidal artery and to a lesser degree by a thalamoperforator from the left posterior communicating artery. Deep venous drainage was through the internal cerebral vein. Planned stereotactic radiosurgery (SRS) was delayed for 3 months due to an unrelated severe knee injury resulting in a lower extremity deep venous thrombosis.

Six months after presentation, MRI and MRA performed for SRS planning did not demonstrate the AVM, or

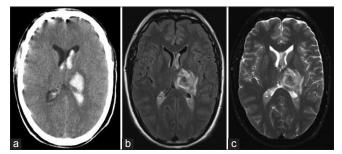


Figure 1: Initial head computed tomography (a: Axial view) and brain magnetic resonance imaging (b: Axial TI-weighted; c: Axial T2-weighted) show a left thalamic intracerebral hemorrhage with ventricular extension and no discernible flow voids to suggest an arteriovenous malformation

the previously seen flow enhancement of the thalamus on MRA. Seven months after initial presentation, DSA and CBCT confirmed the spontaneous angiographic resolution of the AVM [Figure 2c and f] without treatment. The patient was subsequently lost to follow-up.

DISCUSSION

Spontaneous AVM obliteration is a rare, but not unheard of, phenomenon.^[1,2,4,5] Complete regression of an AVM without treatment is not always indicative of good clinical outcomes. Recurrence,^[3,5] *de-novo* AVM formation,^[5] and *de-novo* aneurysm formation^[8] after complete spontaneous regression have been previously described. Although infrequent, these events warrant consideration of follow-up imaging for cases of spontaneous AVM occlusion.

To our knowledge, we report the first case imaged with both DSA and CBCT. Although DSA is considered the gold standard of diagnostic AVM imaging, the modality is at times limited. At initial presentation, the described AVM nidus could not be visualized on DSA due to concurrent hematoma [Figure 2a]. False negative DSA imaging of AVMs at the time of acute hemorrhage has been previously reported.^[10] Rahal and Malek describe the use of CBCT angiography to fully visualize a nidal structure in three AVM cases obscured by hematoma on DSA.^[7] In addition, in this case it is challenging to fully appreciate the 5 mm nidus on DSA [Figure 2b] compared to CBCT [Figure 2d]. High resolution images

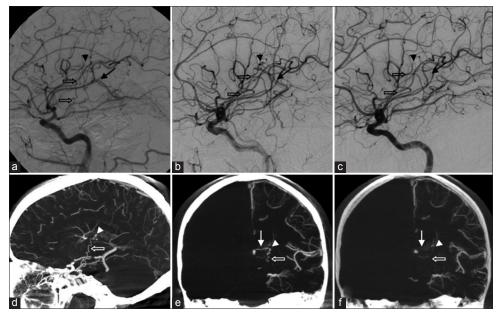


Figure 2: The arteriovenous malformation (AVM) nidus (arrowhead), seen on lateral digital subtraction angiography (a, b and c) and three-dimensional sagittal (d) and coronal (e and f) cone beam computed tomography reconstructions (left carotid injections), is fed by the left anterior choroidal artery (hollow arrow) and a thalamic perforator branch of the left posterior communicating artery (right vertebral injection, not shown), and drained by the left internal cerebral vein (solid arrow). (a) Hematoma obscures the nidus at presentation, (b, d and e) 3 months follow-up reveals the AVM, and (c and f) 7 months follow-up demonstrates complete spontaneous AVM obliteration

of vascular lesions, including micro-AVMs, can be created with CBCT.^[9]

The use of CBCT also facilitates simultaneous identification of feeding arteries and draining veins. Distinguishing the draining vein as the internal cerebral vein from the superficial middle cerebral vein is challenging on DSA due to overlapping vasculature at presentation and diagnosis [Figure 2a and b], compared to CBCT [Figure 2d]. The thalamoperforator from the left posterior communicating feeding artery could only be visualized on the right vertebral injection on DSA. While this artery was not injected on CBCT, in this case, Radvany *et al.* have shown that all four major arterial territories can be injected simultaneously on CBCT.^[6] The potential benefits of CBCT may warrant its use as a supplemental tool in addition to DSA in the confirmation of spontaneous AVM obliteration.

CONCLUSIONS

Patients with complete spontaneous AVM regression should be closely monitored as angiographic obliteration is not always indicative of good outcomes. Future routine use of CBCT as an adjunct to DSA for the diagnosis and follow-up of AVMs may be clinically valuable, particularly for very small AVMs.

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